investigate the left ventricular diastolic function and the factors related in SLE patients compared with healthy controls.

Methods Thirty consecutive female SLE patients without evidence of cardiac disease were underwent standard transthoracic echocardiography, and were compared with 30 age-matched healthy female controls. Patient characteristics, organ damage and laboratory data were retrieved by medical chart review.

Results In SLE patients, indexes of LV diastolic function differed from control group, with reduced early diastolic filling velocity (E), as well as prolongation of the time taken from the maximum E point to baseline, reduced ratio of early to late diastolic flow velocity (E/A), prolonged ratio of E to early diastolic mitral annular velocity (E') (E/E'). However, the differences did not show statistical significance. Anti-Ro antibody positivity was observed in 43% of SLE patients, and it was correlated with higher E/A ratio significantly (1.3±0.4 vs 1.0±0.2, p=0.03). In addition, the SLE patients with hematologic or renal involvement showed more enlarged size of left atrium significantly compared to the patients without any involvement (36±4.3 vs 31±9.2, p=0.01).

Conclusions Although not statistically significant, there was a trend which suggested that patients with SLE have subclinical impaired diastolic function compared with the healthy control. Presence of anti-Ro antibody and systemic organ involvement was related with the diastolic dysfunction markers.

Conclusions Our data demonstrated a role of EDA+FN in the pathogenesis of tubulo-interstitial disease in lupus nephritis.

Background and aims Anti-dsDNA antibody plays a critical role in the pathogenesis of lupus nephritis and contributes to inflammatory and fibrotic processes in the kidney. EDA+ spliced variant of fibronectin (EDA+ FN), normally only weakly expressed, is markedly increased in pathological conditions. We investigated the effect of human polyclonal anti-dsDNA antibodies on the expression of EDA+FN in proximal tubular epithelial cells (PTEC) and the functional consequence.

Methods EDA+ FN expression in human renal biopsies of Class III/IV±V lupus nephritis was assessed by cytochemistry. Cultured PTEC were incubated with control IgG or IgG anti-dsDNA antibodies isolated from lupus nephritis patients for 24 hours and the expression of EDA+FN was investigated. Recombinant human EDA peptide was used to investigate the functional role of EDA+FN in PTEC.

Results The results showed that EDA+FN was absent from normal kidney tissue but was markedly increased in the tubulo-interstitium in lupus nephritis patients. Cultured PTEC constitutively expressed native FN but not EDA+FN. Anti-dsDNA antibodies, compared with serum-free-medium and control IgG, increased EDA+FN expression by 5.8- and 5.6-fold respectively (p<0.05 for both), and the induction was mediated through PI3K and mTOR activation. Exogenous IL-1β and TGF-β1, but not IL-6, IL-8 or MCP-1, induced EDA+FN by 1.8- and 2.3-fold respectively. Recombinant EDA peptide increased native FN, collagen I, laminin and SNAIL expression, but decreased E-cadherin expression, in PTEC.

Conclusions The joint deposited IgG exerts an important role in the development of lupus arthritis lacking of bone destruction, Syk plays a crucial role in lupus IgG-induced arthritis and inhibited osteoclastogenesis. This finding will promote development of effective therapeutic strategy to arthritis in SLE patients.
promoted these effects through the membrane receptor GPER1 located in lipid rafts and that inhibition of lipid rafts and GPER1 suppressed SLE serum-induced skin inflammation and expression of inflammatory molecules.

Conclusions We conclude that oestrogen promotes the development of skin injury induced by SLE serum through the membrane receptor GPER1 and that lipid rafts play an important role in the regulatory effect of GPER1 in SLE skin inflammation.

Results We found that the severity of skin lesion in IL-1 receptor deficient mice and caspase-1 deficient mice was reduced compared with that in wild type mice. IL-1 receptor deficiency suppressed the expression of FcγRI (CD64) and MHC class II (CD74), and increased the level of FcγRII (CD32) induced by lupus serum. IL-1 receptor deficiency also suppressed the lipid raft clustering and IFN-γ in T cells, and reduced IgG internalisation and presentation in macrophage, and decreased expression of MCP-1 and TNFα in monocytes. In addition, TNFα could promote the proliferation of keratinocytes.

Conclusions Our findings indicate that IL-1 plays an important role in skin lesions of lupus erythematosus. This study suggests IL-1 is a therapeutic target in skin lesions of systemic lupus erythematosus.

Background and aims Skin injury is the second most common clinical manifestation in patients with systemic lupus erythematosus (SLE), but its pathogenesis has not been thoroughly elucidated.

Methods Based on skin deposition of IgG in SLE, we studied the features and mechanisms of intradermal IgG-induced skin inflammation.

Results We found that skin inflammation appeared at 3 hour and peaked at 3 d after intradermal injection of lupus IgG. This phenomenon was related to the dose of injected IgG but not to systemic disease activity. The severity of skin inflammation induced by lupus IgG was significantly decreased in mice depleted of monocytes and in mice deficient in TNF-α but not in mice missing mature lymphocytes. Furthermore, lupus IgG promoted the progression of monocyte differentiation to dendritic cells (DCs) and enhanced the expression of TNF-α.

TNF-α was found to stimulate the IgG-induced maturation of DCs and played a major role in the proliferation and activation of keratinocytes.

Conclusions The results also indicate that the deposition of IgG in skin exerts an important role in the pathogenesis of skin injury in patients with SLE; therefore, blocking the IgG/FcR signalling pathway can be a therapeutic target in skin lesions of patients with SLE.

Results We found that the severity of skin lesion in IL-1 receptor deficient mice and caspase-1 deficient mice was reduced compared with that in wild type mice. IL-1 receptor deficiency suppressed the expression of FcγRI (CD64) and MHC class II (CD74), and increased the level of FcγRII (CD32) induced by lupus serum. IL-1 receptor deficiency also suppressed the lipid raft clustering and IFN-γ in T cells, and reduced IgG internalisation and presentation in macrophage, and decreased expression of MCP-1 and TNFα in monocytes. In addition, TNFα could promote the proliferation of keratinocytes.

Conclusions Our findings indicate that IL-1 plays an important role in skin lesions of lupus erythematosus. This study suggests IL-1 is a therapeutic target in skin lesions of systemic lupus erythematosus.